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Inspiratory Efforts During Mechanical Ventilation:

Is There Risk of Barotrauma?

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A growing body of literature suggests that mechanical ventilation can promote lung damage when excessive transpulmonary pressures are applied.¹ Although stretch-mediated lung injury has received much attention,² lung hyperinflation leading to gross barotrauma is also a concern, particularly if lung volume exceeds total lung capacity (TLC). Human lungs are normally prevented from overdistension by the felicitous match between maximal inspiratory muscle pressures (P_{mus}-max) and the pressures required to inflate the respiratory system to its maximal volume (*ie*, TLC). This protective equilibrium may not apply to patients making forceful inspiratory efforts on mechanical ventilation, especially during pressure-control or pressure-support ventilation when applied pressures could conceivably combine with P_{mus}-max to overdistend the lungs.

In this issue of *CHEST* (see page 711), Sinderby et al³ provide observations that make the possibility of involuntary hyperinflation seem less likely. Maximal lung volume (*ie*, TLC) is quite reproducible in trained individuals; repeated inhalations yield TLC values clustering within 5%. What limits TLC in healthy subjects? Early reports^{4–6} that abdominal muscles become active when subjects inhale to TLC, thereby raising the abdominal pressure, led to suggestions that TLC is limited by the reflex action of the expiratory abdominal muscles. In addition, glottal closure during sustained inhalation to TLC led to the conclusion that airway reflexes also limit maximal lung volume.⁶ Later, Mead et al⁷ showed that inhalation to TLC could be performed with or without abdominal muscle contraction, observing substantial abdominal muscle contraction only in untrained subjects. In fact, modest abdominal muscle contraction during active inhalation at TLC has only minimal expiratory effect. The study by Mead et al⁷ led to the current understanding that TLC is limited by a mechanical equilibrium between P_{mus}-max and the elastic recoil of the lung and chest wall (Fig 1). Subsequent demonstration that the diaphragm is maximally active during inhalation at TLC^{8–10} implied that reflexes are not normally important in limiting maximal lung volume.

Of the three mechanical components limiting maximal lung volume, P_{mus}-max is probably the most important. The rationale for this statement is based on the balance of the following three pressures that determine TLC: the passive inward expiratory recoil of the chest wall (about 15 cm H₂O); the inward expiratory elastic recoil of the lung (about 25 cm H₂O); and the inspiratory pressure of the respiratory muscles (40 cm H₂O), which equals the sum of the recoil pressures of the lung and chest wall. It is the slope of these volume-pressure characteristics (called *tangent compliance*) at TLC that determines their importance in limiting maximal

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volume. For example, if the lung were infinitely stiff at TLC, increases in inspiratory muscle force at TLC would not be able to increase volume above TLC, and we would conclude that the stiffness of the lung, not Pmus-max, is the more important determinant of TLC. By contrast, if the lung and the passive chest wall were relatively compliant, small changes in Pmus-max at TLC could have a substantial effect on the volume achieved, and we would say that the passive characteristics of the lung and chest wall were not very important in determining TLC. In fact, the least compliant of the three pressure-volume characteristics is that of Pmus-max (Fig 1), which is relatively flat near TLC,^{9,11} compared with those of the lung¹² and the relaxed chest wall. Thus, because Pmus-max decays so rapidly with increasing volume near TLC, small changes in the recoil pressures of the rib cage and lung would have a relatively small effect on TLC. The importance of Pmus-max in determining TLC is supported by experiments¹³ showing that partial curarization reduces TLC substantially at doses that produce only modest reductions in inspiratory muscle force at resting lung volume.

What has the article by Sinderby et al³ contributed to this understanding? Using neurally adjusted ventilatory assist (NAVA), they explored the interactions between mechanical ventilation and respiratory muscle action in healthy subjects making maximal inhalations. NAVA applies pressure in proportion to electromyogram activity in the diaphragm in an attempt to maximize comfort, which is somewhat similar to proportional assist ventilation (PAV). As they increased ventilatory assist from zero to a level that provided all of the pressure required for lung inflation, they observed a decrease in diaphragmatic activation such that the sum of inspiratory Pmus-max and ventilator pressures continued to exactly match the pressure required for lung inflation. When the subjects voluntarily made maximal inspiratory efforts, the maximal diaphragm activity was similarly reduced to a level at which the inspiratory capacity remained near the unassisted values, and, despite airway pressures that were equal to those normally supplied by Pmus-max, lung volume at TLC remained nearly unchanged. These observations suggest that in healthy subjects diaphragmatic activation is limited, perhaps by reflex, to maintain maximal lung volume constant in the face of externally applied inspiratory pressures. While this reflex is not the same as that proposed by early investigators, it does suggest that maximal lung volumes will not be exceeded during NAVA in patients who are also using their inspiratory muscles. Two important caveats need to be mentioned. These studies were conducted in healthy subjects making voluntary inspiratory efforts, not in dyspneic patients with injured lungs making involuntary inhalations. Heterogeneously diseased lungs may experience high shear forces at relatively low transpulmonary pressures, particularly at the junctions of normal and abnormal lung tissue. Furthermore, these experiments suggest that inspiratory muscle actions are limited at high lung volumes, but forceful inspiratory efforts at low lung volumes could also produce regional overexpansion in a heterogeneously diseased lung.

As in PAV, NAVA relies on the concept that ICU patients should determine their own respiratory rate and tidal volume. NAVA has a potential advantage over PAV in that inflation is triggered at the start of the inspiratory effort (even with auto-positive end-expiratory pressure), rather than requiring the patient to initiate airflow. On the other hand, NAVA requires the continuous assessment of electromyogram activity in the diaphragm, a signal that may be difficult to record reliably for prolonged periods. For the physiologist, this article provides support for a possible inhibitory reflex that can reduce diaphragm activation to prevent lung overexpansion. For the clinician, questions remain about when NAVA should be used and whether this technique improves patient/ventilator synchrony, safety, and comfort.

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Figure 1.

Elastic recoil pressure of the lung and relaxed chest wall and the Pmus-max from residual volume to TLC. At resting end-exhalation (*ie*, functional residual capacity (FRC)), the elastic recoil pressures of the lung and chest wall are equal and opposite. Near TLC, Pmus-max diminishes very rapidly with increasing volume, largely determining the upper limit of the lung volume.